

***Neurocardiogenic Syncope in the Intercollegiate Athlete:
An Examination of Problems and Solutions***

An Honors Thesis (HONRS 499)

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Abstract

Neurocardiogenic Syncope, a condition common in young people, is becoming more recognized in the Athletic Training profession. The purpose of this discussion is to delve into this condition and provide basic knowledge to help the Athletic Trainer manage athletes with this condition. The information will be divided into two sections. First, the examination will describe Neurocardiogenic Syncope, its presentation, anatomical and physiological causes, warning signs and techniques for diagnosis. It will also examine the decreased quality of both daily living and performance due to this condition. The last part of the first section will attempt to summarize numerous treatment options available to the athlete with Neurocardiogenic Syncope. The second section will present a case study of an intercollegiate athlete with this condition and its effect on the athlete's performance and overall well being. It is hoped that this examination can bring to light a new and challenging condition that is becoming more frequently recognized in the Athletic Training profession and to offer suggestions to make the problem more manageable. It is also my goal that the information in this paper can be used to improve the quality of health care that Athletic Trainers can provide for the athlete with Neurocardiogenic Syncope.

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Introduction

In many medical professions, ideas and facts are always changing. The field of Sports Medicine is no different. As athletes and athletic competition grow stronger every day, the Athletic Training profession must stay abreast of each new condition, illness or injury that can slow the athlete down. One condition that has recently emerged into the spotlight is fainting, or syncope. Syncope is a problem in today's athletic setting because it can be indicative of many underlying conditions. It can be a signal of heart problems, nutritional problems and several other illnesses. Today's Certified Athletic Trainer must be aware of the causes of syncope, the proper treatment, and the proper actions to perform in an emergency. It is also important that the Athletic Trainer be knowledgeable enough about syncopal conditions to be able to educate the athlete.

One particular type of syncope that is beginning to be more recognized in the athletic arena is Neurocardiogenic Syncope. This is a benign condition that stems from an orthostatic imbalance between the brain and the heart. More and more athletes are learning proper treatment for the condition, however, and are able to continue participation. This has a great deal of connotation for the Athletic Trainer. The Athletic Trainer must now educate himself or herself on the causes, treatments, and preventative measures that need to be taken when dealing with athletes who have neurocardiogenic syncope. This recognition and understanding is especially important on the intercollegiate level, due to the common pressures present for these elite athletes. Especially when working on the Division I level, the Athletic Trainer is responsible for keeping athletes playing, if possible. Syncope can make that task challenging. Syncope contains a broad spectrum of signs and symptoms, treatments, nutritional factors and

much more. The purpose of this examination and the following case study is to explain the presentation, recognition, anatomical and physiological concerns, prodrome, diagnostic techniques and testing, effects of daily living, preparticipation screening and treatment options associated with neurocardiogenic syncope. It will also examine the implications this information has for the intercollegiate athlete.

Presentation and Recognition Factors

For proper and adequate understanding of the remaining content of this examination of neurocardiogenic syncope, a few concepts must be defined and clarified. First, as Basilico notes, it is important to realize that physicians, coaches, and Athletic Trainers are used to seeing athletes at their peak levels of fitness and it is hard to believe that any underlying cardiac abnormality could exist in any of these elite young men and women.¹ When an athlete presents with syncope, however, the possibility of a cardiac abnormality must be investigated regardless of how the examiner may feel about the health of the athlete. So, just what is syncope? The medical definition for syncope is a sudden and transient loss of consciousness and postural tone with spontaneous recovery.² In lay terms, syncope is often called “fainting” or “passing out”. A related condition is presyncope, which involves the feeling that one is going to lose consciousness without ever fully reaching an unconscious state. There are many different types of syncope, as will be discussed later, but the one focused on in this paper will be Neurocardiogenic Syncope. This condition has many names, including Neurally Mediated Hypotension, Vasodepressor Syncope, Vasovagal Attack, and Vasovagal Syncope.³ No matter which name is used, however, the condition is a common disorder of autonomic cardiovascular

regulation, which presents itself as syncope or presyncope.³ The diagnosis of this condition in the general population is fairly new, with its appearance first being documented in 1932 by Sir Thomas Lewis.^{4,5} Lewis used the name vasovagal syncope to describe the vasovagal inhibition that occurs during a syncopal episode.⁵

Syncope is more common in the general population than some may realize. As Shen reports, syncope will occur at least once in the lifetime of about one third of the general population.⁶ It is quite possible that many of these syncopal episodes may be due to neurocardiogenic syncope. The possibility of a syncopal episode, however, is present in all humans.⁴ It is for this reason, as Barbey states, that neurocardiogenic syncope must not be seen as a disease, but rather as an inappropriate reaction or reflex to a stress placed on the body.⁴ As Calkins states, most research shows that neurocardiogenic syncope is most frequently seen in young adults. The condition is also more commonly seen in women than in men.³ Syncope can often occur in the athletic arena, however, and the Athletic Trainer must be prepared to recognize the condition. In fact, this condition is the most common reason for syncopal episodes in the athletic setting.⁷ This recognition is not easy, however, and can be confused with many other conditions.

The first step in recognizing neurocardiogenic syncope is recognizing the condition's common patterns of presentation. In the athletic arena, an athlete experiencing a syncopal episode during play or on the sidelines creates a dramatic scene.⁷ This can be especially true at the high school level, where a young child is being watched by worried parents. In young athletes, syncopal episodes can be confused with Sudden Cardiac Death (SCD). SCD occurs when an athlete collapses and dies suddenly due to a heart abnormality. SCD is more well known than syncope, so the unknowing spectator

may automatically assume that the syncopal athlete is in grave danger during a syncopal episode.⁸ The Athletic Trainer and Coach must be prepared to deal with the syncopal episode and must be prepared to field questions to those who may overreact to the episode. Syncope can be dangerous, however, because of the high risk of secondary injury to the athlete. Injuries can occur if the athlete falls down during a syncopal episode or if the athlete is playing in a high contact sport.⁸ There is also a danger of an episode occurring while the athlete is away from the field. Driving, for example, can be risky if the athlete has a severe case of neurocardiogenic syncope that is not controlled.

One problem with neurocardiogenic syncope in the athletic setting is the variety of times and places in which an episode may occur. As Calkins, Seifert and Morady state, syncope may occur in several situations. It may present 1) during or after exercise; 2) in trained and untrained athletes; 3) in patients who experience neurocardiogenic syncope not related to exercise; 4) not related to an exercise stress test; or 5) with or without a prodrome (or warning signs).⁹ In other words, this condition can be seen at almost any time, any where, in anyone. Many times neurocardiogenic syncope is related to emotional distress, the sight of blood, pain or injury. Some researchers have also found that athletes will experience episodes of syncope in clusters. The attacks will become more prevalent for a time, and then will die off for a time.¹⁰ It is a “come and go” type problem. This makes it even harder to predict episodes and times when the athlete will be most affected.

If the athlete and the Athletic Trainer learn to recognize attacks, many of them can be prevented. For example, attacks almost always present when the thorax is in a vertical position.⁴ In other words, the athlete is usually standing up. In many cases, a full

syncopal episode can be avoided if the athlete is placed in a supine position on a level surface. As Barbey notes, however, many times neurocardiogenic syncope attacks present at times when it is either impossible or socially unacceptable to assume this corrective position.⁴ This poses obvious problems; the athlete not only has an episode but is also highly visible in social surroundings, making the situation difficult and uncomfortable for everyone involved. The more the athlete can learn to avoid these situations, the better he or she will be able to adjust to the syncopal episodes as they occur.

Another issue that must be addressed before looking at the specific physiological details of neurocardiogenic syncope are the different causes of general syncope. Syncope falls into three general categories: 1) Cardiovascular; 2) Noncardiovascular; and 3) Unexplained.⁶ Cardiovascular Syncope involves specific problems with the cardiovascular systems, such as heart arrhythmia, cardiomyopathy, congenital heart disease and other specific heart problems. Athletes or patients with cardiovascular (also known as cardiac) based syncopal conditions have a potentially life-threatening condition and should not continue participation. Noncardiovascular Syncope, on the other hand, includes conditions like neurocardiogenic syncope. It is not a problem with the heart itself, but rather a series of other malfunctions that lead to syncopal episodes. This type of syncope is much less dangerous and not known to be fatal in most cases. Unexplained Syncope contains conditions that can not be diagnosed despite diagnostic testing. In some cases, there is a psychological factor contributing to the syncopal episodes.¹¹ It has been noted that patients under the age of 45 with psychologically based syncope usually

associate the episodes with other symptoms, like headaches.¹¹ In any case, constant efforts must be made to attempt to find a diagnosis for unexplained cases of syncope.

Anatomical and Physiological Concerns

The physiological process behind each episode of neurocardiogenic syncope is very complicated and involves several body structures. A basic review of the anatomy and physiology involved as presented by Tortora and Grabowski helps with understanding of these concepts. First, refer to the diagram of the human heart in Appendix A. This diagram shows the movement of blood through the heart and lungs in a normal human being. In a normal human, blood is carried to the right atrium from the veins. It is then pumped to the right ventricle and then on to the lungs. Oxygenated blood returns to the left atrium from the lungs and is pumped into the left ventricle. It is the left ventricle which is responsible for pumping the oxygenated blood to the rest of the body. If all parts of the cardiovascular system are functioning properly, there is always ample blood returning to the heart from the extremities to be pumped through the heart to begin the whole process again. The quantity of blood returning to the heart from the body is termed venous return. In turn, the amount of blood the heart pumps back out to the extremities is termed cardiac output. In short, when venous return decreases, the cardiac output will also decrease. The body will have to compensate, if possible, by adjusting heart rate until venous return can increase again.¹²

In the average person, there is some amount of pressure in the vascular system at all times. This pressure on the walls of the blood vessels is generated by the contraction of the ventricles and is termed blood pressure.¹² Blood pressure varies according to the

amount of stress the body is under, due to the increase and decrease in heart rate and changes in vascular constriction levels experienced when stress levels change. For example, during exercise, the heart rate will increase to meet the incessant demand for oxygen from the body tissues. Blood pressure will increase as well, due to the increased rate of contraction of the ventricles. The blood pressure of the average adult at rest is 120/80mmHg.¹² The number 120 depicts systole, or the contraction of the ventricles and the 80 represents diastole, or the relaxation of the ventricles.¹² This pressure will fluctuate during normal cycles of the day and will increase when the body is put under stress, such as exercise or perceived psychological stress.

When the normal human assumes an upright posture, there are several changes in the cardiovascular system. First of all, when assuming an upright position, a great deal of blood from the thoracic cavity displaces to and pools in the lower extremities.² For the average human, this amounts to between 300mL to 800mL of blood that is displaced from the vital organs in the thoracic cavity.¹⁴ The body must be able to compensate for this sudden movement of blood to the extremities. As Grubb and Karas note, under normal circumstances, the body can adjust to this change quickly by slightly increasing heart rate to return the blood to the heart. There is little change noticed in the blood pressure, because the adjustment is made so quickly by the body.² In fact, the body can usually achieve orthostatic balance (a balance in blood pressure and return to normal heart rate) within 60 seconds.¹³ The person who has performed the task of standing up has no idea that the body has just made this adjustment. The body finds its baseline condition and returns to it quickly without the details of the change ever reaching the consciousness of the mind.

An increase or decrease in heart rate and changes in blood pressure are not just “achieved.” There are several body structures that make these adjustments possible. First, the heart is constantly under the influence of two types of fibers, which both stem from the medulla oblongata in the brain. One set of these fibers causes the heart rate to speed up (or stimulate) and is termed the sympathetic branch.^{12,13} The other set functions as an opposing force to the sympathetic fibers. These fibers are termed the parasympathetic fibers and are responsible for slowing down (or inhibiting) the heart rate.¹²⁻¹³ Both the sympathetic and parasympathetic fibers are under autonomic control and are therefore involuntary.¹² These two opposing sets of fibers are constantly in balance and will adjust to any body changes to remain so.¹² It is this balance that controls the heart rate according to the needs of the body.

The heart also has two groups of nerve cells that help maintain normal blood pressure. As Tortora and Grabowski note, the first of these groups are termed baroreceptors. The baroreceptors respond to changes in the amount of pressure, or stretch, that is being placed on the blood vessels. These receptors constantly monitor the amount of pressure being exerted in the arteries, veins and the right atrium. If there is a change in pressure, the baroreceptors alert the brain so the proper actions can be taken to adjust the body’s overall blood pressure. If the blood pressure is low, for example, the baroreceptors will notify the brain so the vessels will vasoconstrict to increase pressure. The second group of receptors used to control blood pressure is chemoreceptors. These receptors monitor the chemical balance in the blood and send messages for it to be adjusted accordingly. If more oxygen is needed, as in the case of exercise, the chemoreceptors will begin the chain of commands to make this adjustment.¹²

Normally, when a human assumes an upright posture, there is an increase in heart rate caused by increased sympathetic activity and peripheral vascular resistance brought about by the changes noted by baroreceptors and chemoreceptors.² All parts of the body should get adequate amounts of blood and be able to function normally. In the patient with neurocardiogenic syncope, however, this is not the case. Syncope is actually caused by a decrease in perfusion of blood in the brain.⁷ If this decrease in perfusion continues for approximately 8 to 10 seconds, the areas of the brain that are responsible for maintaining consciousness do not get an adequate blood supply and the result is unconsciousness.^{7,11} When the neurocardiogenic syncopal athlete displaces blood to the lower extremities, it pools just like in a normal person.¹⁴ The syncopal athlete, however, does not compensate for the sudden movement of blood. This misstep causes a decrease in venous return.¹⁴ Normally, this decrease in venous return should cause an increase in heart rate and systemic vasoconstriction of the peripheral blood vessels.¹² The brain of the syncopal athlete, however, will get the messages confused. She will experience a decrease in heart rate, termed bradycardia, and systemic vasodilation.¹⁴ Both of these factors lead to a decrease in cardiac output, which in turn leads to decreased perfusion in the brain and results in syncopal or presyncopal episodes.³

At this time, researchers are still trying to discover how the brain confuses the messages necessary to keep perfusion at appropriate levels. Tortora and Grabowski state that beta adrenergic receptors in the skeletal muscles and the heart cause the sympathetic message from the brain to stimulate vasodilation rather than vasoconstriction.¹² For this reason, many treatments involve using beta-blocking drugs to attempt to prevent this reaction.² This is a challenging question, however, because the body of the syncopal

athlete is performing the exact opposite reactions of what it should be.⁷ Many athletes,

for example, will say that they feel their heart is not “keeping up with them.” It is the bradycardia that makes the athlete feel this way.⁷ They feel that the heart is simply not pumping quickly enough to deliver the body cells the oxygen they need to function. When the athlete is engaging in physical activity, this can be a very uncomfortable feeling, due to the increased need of oxygen from the cells. As the heart continues to beat slowly, however, the increase in demand for oxygen causes the heart to increase the strength of ventricular contractions, attempting to fill the needs of the cells.¹⁴ This increases the discomfort that the athlete experiences. Not only is her heart beating slowly, it feels as if it is pounding inside her chest. No matter how hard the heart pounds, however, it is unsuccessfully trying to pump blood from an empty chamber.¹⁵ Not until the body is able to increase venous return will the heart once again become efficient.

As for the brain, there is no other choice but to shut the body down to a state of unconsciousness. It is not that the brain is in immediate danger, but is taking protective measures before the situation gets any worse. Each structure of the brain needs a great deal of oxygen to work properly.¹⁶ When it does not have the blood it needs, it has to shut down the functions that are less important so the areas that need blood to keep the body alive can continue to function.¹⁶ In this case, consciousness is controlled by the reticular activating formation. When this structure does not receive a proper amount of blood, it makes its move to protect the body. Unfortunately, evolution has placed the human brain at a disadvantage. Since athletes are almost constantly in a vertical position, the blood has to be pumped against gravity to reach the most important organ in the body.⁴ If all functions are not performed exactly as needed, as in the case of the syncopal

athlete, that precious blood supply is all too quickly compromised.⁴ This concept makes it easy for both the athlete and the Athletic Trainer to understand that the athlete should always lay down on their back at the first signs of an attack.⁴ This position puts the brain in a more advantageous location so the heart will no longer be forced to work against gravity.⁴ If the attack is recognized quickly and the proper position is assumed, a full syncopal episode may be avoided.

The Prodrome

Since avoiding full syncopal episodes is the goal with any athlete that has neurocardiogenic syncope, the athlete, Athletic Trainer, coach and any of those responsible for the athlete must make themselves aware of the signs and symptoms of an attack. In most cases, there are a set of signs and symptoms that occur before each episode.⁶ These signs and symptoms include (but are not necessarily limited to) pallor, weakness, lightheadedness, yawning, nausea, diaphoresis (excessive sweating), hyperventilation, blurred vision, and impaired hearing.⁶ Some athletes will also complain of a numb, tingling feeling in their hands. Collectively, these warning signs of an attack are termed the prodrome.⁷ It is important to note that each individual experiences his or her own set of symptoms before each attack.⁴ The prodrome that one athlete feels will not necessarily be the same symptoms that the next athlete will experience.⁴ It is important that the athlete and the Athletic Trainer keep the lines of communication open as the athlete gains experience in recognizing the prodrome. Once the athlete has a couple episodes, she will see a pattern in how she feels before each attack and will be able to recognize those symptoms.

Some research has supported the idea that syncopal episodes are preceded by an aura sensation. An aura is a short perceptive experience with somatosensory, autonomic, psychic or compound content that can occur as part of the prodrome.¹⁷ In other words the athlete simply “feels” as if something in the body simply isn’t right. For example, the athlete may feel some palpitations, which makes them feel as if there is something wrong.¹ Some athletes will state that they feel somewhat sick, weak or uneasy several minutes before the presyncopal or syncopal episode begins.¹⁷ Some researchers ignore the aura phenomenon and consider the idea phenomenon more of a “psychic” reaction, but other researchers have found evidence that this phenomenon actually may be experienced during the initial moments of a presyncopal episode.¹⁷ Interestingly, this type of phenomenon is noted and accepted as an identifying symptom of epileptic seizures.¹⁷ It has been thought by some researchers that the absence or differences in the types of aura experienced between neurocardiogenic syncope patients and those with ictal disorders could be a key to differentially diagnosing syncope, but this has not yet proven to be affective.¹⁷

Neurocardiogenic attacks do not always occur with a prodrome, however.⁷ Some syncopal episodes, unfortunately, will have no warning signs. This type of syncopal attack is termed Malignant Vasovagal Syndrome.⁴ Some athletes will experience neurocardiogenic syncope attacks with a prodrome every time, some with no prodrome each time and some with a combination of the two. Malignant attacks are very dangerous. Since there is no prodrome, the athlete has no idea the attack is coming. The athlete could be standing still, competing, or even driving when the attack manifests. Because of these risks, there is no choice but to treat the patient immediately. Every

method possible must be attempted to prevent these malignant attacks. Continuing with athletic participation would be considered very risky for the athlete with malignant vasovagal syndrome. Malignant attacks could also be a warning sign that there are causes other than neurocardiogenic syncope leading to unconsciousness and should be investigated. If the majority of the athlete's episodes are non-malignant, however, the risk is much less. The athlete and the Athletic Trainer must simply attempt to prevent as many episodes as possible.

Techniques for Diagnosis

From the physician's standpoint, neurocardiogenic syncope can be very hard to diagnose. One reason diagnosis is frustrating is due to the fact that syncope can be both a symptom and a sign.² In other words, it can be caused by other things, or it can be causing other problems. For example, an athlete who presents with syncopal episodes could have a heart condition that is causing the problem. In that case, syncope is a symptom. There are also several precipitating factors of which syncope is a sign. Some of these factors include anemia, dehydration, hunger, recent illness, physical exhaustion and too much time trapped in crowded, poorly ventilated areas.¹⁸ Diagnosis of neurocardiogenic syncope is also difficult because there are so many possible causes for the condition.¹⁴ All possibilities must be explored to ensure the safety of the athlete during competition. Unfortunately, in up to 50% of patients that present with cases of syncope, no actual diagnosis can be made concerning the cause.¹⁹ These cases of syncope fall under the "unexplained" category mentioned earlier. Finding a correct

diagnosis in athletes is particularly important. If the athlete feels at all unsure about her condition, she will not feel comfortable or safe returning to competition.³

There are several possibilities pertaining to the cause of syncope. The athlete could be experiencing anything from neurocardiogenic syncope to drop attacks, heart malfunctions, vertigo, dehydration or even seizures.⁴ The first objective in evaluating any case of syncope, however, is to rule out an underlying cardiac disorder.¹¹ Cardiac disorders are the most dangerous cause of syncope and must be identified as quickly as possible. The first and most vital step in the diagnosis process is the history. The initial history, when combined with a physical examination has been found to identify the cause of syncope anywhere from 40% to 85% of the time.^{6,11} Aspects of the history that should point the physician in the direction of a neurocardiogenic syncope diagnosis include young age; no previous history of cardiac disease or complications; presence of a prodrome; and presence of post-syncopal fatigue.³ The physician needs to be diligent when asking questions about diet, hydration, activity levels, previous history of syncopal episodes and family cardiac history. It is also very important that the physician ask the athlete specific details about all the medications that she is taking.⁶ In the college athlete, it is important to note that lack of sleep and excessive alcohol intake can predispose an athlete to neurocardiogenic syncope.¹¹ At times, an outside observer, such as a parent or the athlete's Athletic Trainer can be helpful during the history portion of an examination.²⁰ The outside observer can remember aspects of each episode that the athlete may not remember or may not be able to view because he or she is unconscious.²⁰

Diagnostic Testing

The next step in diagnosing neurocardiogenic syncope is conducting a comprehensive physical examination. This should include a complete thorough neurologic and cardiovascular evaluation.²⁰ It should look for signs of heart malfunction, including orthostatic hypotension or any congenital heart conditions.²⁰ Orthostatic hypotension is a fall of systolic blood pressure greater than 20mmHg or diastolic pressure greater than 10mmHg after the athlete has assumed and maintained a standing position for at least three minutes.¹⁵ As stated before, the physician must be on the lookout for any sign or symptom that would lead to a cardiac abnormality. It is also helpful in the physical examination if the physician is aware that neurocardiogenic syncope commonly occurs in three patterns, each of which has its own physiological marker.¹⁸ The three patterns are: 1) vasodepressor type, marked by hypotension; 2) cardioinhibitory type, marked by bradycardia; and 3) a combination of both of these types.¹⁸ Knowledge of these three patterns and their physiologic markers, when combined with a good history, can help the physician come to a better diagnosis of the condition.

Although ruling out cardiac abnormalities should be first priority when examining the syncopal patient, it is also important to rule out ictal disorders, also known as seizures.¹⁶ Seizures present themselves in many forms, from an absence seizure to grand mal seizures. Some seizures, especially the grand mal seizure and some petit mal seizures, are characterized by involuntary movement of the body.¹⁶ During some neurocardiogenic episodes, these same seizure-like movements can be noted in the athlete.²¹ If these movements are described by the observer questioned in the athlete's

history, they must be examined so seizures can be ruled out. One key to differentiating a seizure from a neurocardiogenic syncope attack is the experiencing of post-seizure confusion.¹¹ After a syncopal episode, the athlete should not experience confusion.⁶ This is not to say that there will not be a period of fatigue, because that is very common in neurocardiogenic syncope.²¹ After a seizure, however, the patient experiences a great deal of confusion and has no sense of what has just happened to them and why. An athlete who has experienced a syncopal episode, however, should be tired, but alert and aware.

The next logical step in the diagnosis of neurocardiogenic syncope is the utilization of diagnostic testing. There are several different types of diagnostic tests and devices that can be used when diagnosing neurocardiogenic syncope. However, because many of these tests can be unpredictable and at time unreliable, the Athletic Trainer and team physician must always be on the lookout for differential signs and symptoms.¹ The first test used in almost anyone suspected of having a cardiac abnormality is the ECG, or electrocardiogram (also known as EKG). An ECG consists of electrodes, termed leads (the most commonly used is the 12-lead ECG) and a recorder to read the input from the electrodes.¹² These leads are attached to the skin and can pick up electrical currents given off by the heart.¹² These currents are then recorded and printed out by the recorder. The printout consists of both peaks and valleys, termed waves, that are created as the heart beats.¹² An example of what a normal heart beat recording should look like is pictured in Appendix B. As you can see, the large peak on the ECG represents the QRS Complex, which depicts the ventricular depolarization during contraction.¹² The smaller peak after the QRS Complex represents the T Complex and ventricular repolarization

during relaxation of the ventricles.¹² The distance from Q to T on an ECG is important, because if this interval is longer than normal, this indicates a cardiac problem. This problem is termed Long Q-T Syndrome.¹² An athlete with neurocardiogenic syncope, however, will usually have a normal ECG. This test, therefore, is necessary to rule out cardiac abnormality, but does not help much in the specific diagnosis of neurocardiogenic syncope.²⁰

Another test commonly used in any patient suspected of cardiac deficiency is an echocardiogram.²⁰ An echocardiogram uses an ultrasound machine to view the heart beating inside the chest.²⁰ This test is very useful when trying to determine if blood is not flowing through the heart normally for one reason or another. This test can show the physician if the valves are not functioning properly, if there is a deformation of the heart, or any other defect that would cause the heart to not function properly.²⁰ The results of this test are video taped for review by a cardiologist. As is the case with an ECG, this test is essential during evaluation of any patient with a suspected heart abnormality. It does not, however, show much evidence that would help diagnose neurocardiogenic syncope.²⁰ It is a great tool, however, to be sure there are no defects of the heart muscle or it's valves.

As Hammill explains, a third test used in the diagnosis of neurocardiogenic syncope is the Exercise Stress Test. This test is mandated if the athlete experiences syncopal episodes during or after exercise. The test includes an ECG that is taken as the athlete is stressed physically on a treadmill. It also includes repeated blood pressure checks throughout the duration of test and during the period of recovery. One important aspect to remember about the exercise stress test is that the athlete must be monitored at

all times. If the athlete should begin to feel faint, the test must be stopped to avoid injury. The athlete should be monitored not only during the activity but during the entire recovery period, watching for signs and symptoms of syncope.²⁰ What is interesting to note about this test, however, is that it does not reproduce the signs and symptoms of neurocardiogenic syncope in most athletes.³ This test, therefore, does not tell the physician or the cardiologist anything about the cause of the patient's syncope. In many cases, an athlete will be tested in this manner, considered ok, and the cause of syncope will go unexplained unless the athlete is unsatisfied and returns for further examination.

There are several other tests that are geared more specifically to diagnosing the syncopal patient who has a noncardiac-based syncopal condition. One of these tests is the ambulatory ECG.²⁰ The ambulatory ECG allows monitoring of the heart throughout the changes of a twenty four hour period.²⁰ These are conducted with similar equipment that is used by a normal ECG, but without all the bulky equipment that can only be used in a hospital environment.²⁰ The data is simply recorded and printed out at the end of the twenty-four hour period.²⁰ The same limitations would apply to these types of devices that would apply to an regular ECG, however. Unless the athlete has an episode during the twenty-four hour period that the ambulatory ECG is worn, there are no conclusive results from this test. They are useful in learning the normal heart patterns that the athlete experiences in a normal day.²⁰ It can also monitor the effects of standing up, walking around and even athletic competition while the athlete participates.²⁰

Event recorders, as Hammill explains, differ from the ECG and the ambulatory ECG in one major way. The event recorder is only activated when the patient feels it necessary to record the activity of the heart, as during a syncopal episode. These devices

are only capable of recording small amounts of data and storing them to be printed off later. Event recorders, also known as memory loop recorders, have electrodes that are worn by the patient at all times, except when showering. They are helpful in recording episodes that occur on an infrequent basis. One of the latest advances in event recorder technology, however, is the invention of implantable loop recorders. These devices are implanted subcutaneously in the upper left chest and are as small or smaller than a credit card. A “beeper” that the athlete carries with her at all times activates the device. The major advantage to these new implantable recorders is that the athlete can go anywhere and do anything while this device is in the chest. Activities like showering are not a problem and the athlete can activate the recorder at any time. The device can also be left in the chest for a prolonged period of time.²⁰

All of the above tests and devices are good to rule out cardiac conditions that would cause syncope. There is a test, however, that is specifically designed to diagnose neurocardiogenic syncope in its true form. This diagnostic test is termed the tilt-table test. The most important aspect of the tilt-table test is that it can actually reproduce the symptoms of neurocardiogenic syncope in the laboratory setting.² In fact, tilt-table testing is just like spontaneous syncope because the same prodrome can be experienced in both instances.² This reproduction of both the prodrome and the episode give the clinician a chance to see what actually happens to the athlete during a syncopal episode.¹⁴ Tilt-table testing requires the athlete to fast overnight before performing the test.¹⁴ The athlete is placed supine on a table with a weight-bearing footboard to help support the body.¹⁴ The table is then slowly tilted to a certain angle and held there until the athlete experiences an syncopal episode.¹⁴ A picture of this procedure can be found in Appendix

C. During the duration of the test, the athlete's blood pressure, ECG and sensations are all being monitored. You will notice if you look at Figure 2 in Appendix C that the heart can be seen slowing into bradycardia and in most patients will even reach a period of asystole.⁵ The amount of tilt of the table is controversial. The American College of Cardiology recommends tilting to an angle of 60 to 80 degrees for 45 minutes.⁴ At times, an intravenous infusion of isoproterenol is used to help provoke the syncopal episode if a first tilt is unsuccessful.¹⁴

There are five broad groups of reactions to tilt-table testing as researched by Grubb, Karas and Kimmel. The five reactions are as follows:

1. Classic Neurocardiogenic Response: A sudden drop in blood pressure followed by a decrease in heart rate.
2. Dysautonomic Response: A gradual decrease in blood pressure until hypotension causes unconsciousness.
3. Postural Orthostatic Tachycardia Syndrome: Low peripheral vascular resistance that the body attempts to correct with tachycardia.
4. Cerebral Syncope: A rare condition that involves vasoconstriction in the brain only.
5. Psychogenic or Psychosomatic Response: No change in blood pressure or heart rate. The patient usually has a psychiatric disorder in this case.

These reactions to tilt-table testing can give the cardiologist clues to the exact mechanism causing the athlete's syncopal episodes.^{2,14}

Tilt-table testing, like any other test in medical examination, is not perfect. There is a possibility that the athlete will show a false-positive or even a false-negative reaction

to tilting. There are several reasons for a false reporting test. First of all, the test may be performed improperly. Research has shown that if the patient is tilted less than 60 degrees, she may not show symptoms because the tilt was not steep enough.⁵ The athlete could also lose consciousness simply from fear or just from the tilt of the table without having any underlying condition. Interestingly enough, highly trained athletes will sometimes have a false-positive reaction to the test.⁷ It is important to follow up with these patients a couple of times to be sure the test was positive. This can be hard, however, because the test itself is an unpleasant experience, especially if the athlete experiences syncope.²² Most athletes that experience syncope during vigorous exercise will have an abnormal reaction (or positive result) to the tilt-test, however, making it a very effective diagnostic tool.⁹

Effects on Daily Living/Participation

Once the physician and the cardiologist have administered all applicable diagnostic tests and have ruled out all other possibilities, they will come to the conclusion that the athlete has neurocardiogenic syncope. The most important thing for the athlete to remember at this time is that this condition is NOT life threatening!³ In fact, According to physicians, it is safe for athletes with exercise-related neurocardiogenic syncope to continue to participate.⁹ What the athlete, coach and Athletic Trainer all need to be concerned with, however, is the risk of secondary injury during episodes. In many cases, the athlete will feel a prodrome, but will continue to push themselves due to internal competitive drive or to keep their spot on the team. This is often true in collegiate athletes. If the athlete continues to ignore the signs of an episode, however, they are more

likely to faint while running down the court or field. These falls can become dangerous, because they can result in trauma to the face, skull, and extremities.¹⁴ Not only is there a risk when athletes are on the playing surface, but there are dangers of episodes and injury when in the workplace or when driving a vehicle.²¹ These dangers must be reviewed and understood by the athlete as soon as the diagnosis is made.²¹

In all cases of neurocardiogenic syncope, it is important that the physician address the subject of driving a motor vehicle. The decision to allow an athlete with neurocardiogenic syncope to drive is a complicated matter.²³ According to Miles, there are several questions that the physician needs to ask both himself and the athlete. Some of these questions are: What is the risk of harming other drivers? What level of risk is appropriate in today's society? How severe are the events and how long before the event does the prodrome begin?²⁹ When asking these questions, it is important that the physician remember that the goal of reaching zero risk for both the athlete and other drivers is unobtainable.²⁹ There is no possible way, even with treatment that the neurocardiogenic syncope patient can be 100% safe when driving. The athlete and the physician need to assess the risk and decide if driving is worth the trouble.²⁹ The same idea applies when allowing the athlete to participate in high contact/high risk sports. There would be more risk, for example, to let the athlete play football than to play tennis. The table in Appendix D gives examples of the possible risk associated with many popular sports.

Although neurocardiogenic syncope is not life threatening and does not cause a great deal of physical impairment, there is a great deal of psychological impairment that the athlete (or any normal patient with the condition) must deal with.¹⁹ Syncope can

often get in the way of a normal lifestyle. In fact, some studies have shown that up to seventy-six percent of subjects state that syncope interferes with their activities of daily living.¹⁹ The athlete constantly has a fear of having a syncopal event looming just above them. This constant worrying about the condition leads to a great deal of morbidity.¹⁹ Morbidity describes how limiting the illness or condition is to daily life, not how likely one is to die from the condition (mortality). In fact, this condition has a very low mortality rate, but a high morbidity rate.⁸ It is the fear of the episode that makes living so hard.⁸ Imagine walking around being afraid to participate in any activity that may stress the body because you fear syncope. It is hard to imagine. In fact, some studies show that even the physician can not understand how impaired the syncopal athlete really feels.⁸ This lack of empathy probably stems from the point of view that the physician uses compared to the athlete. The physician tends to see mortality only, whereas the athlete will usually only see the morbidity.¹⁹ Appendix E contains a functional status questionnaire that is commonly used to assess the impairment of patients.

Preparticipation Screening

In the athletic arena, the most effective solution for problems presented by neurocardiogenic syncope is prevention and proper screening for the condition. In many cases, athletes with neurocardiogenic syncope will not make it to the elite intercollegiate level because they are “weeded out” in the recruiting process. It is important, however, to screen all athletes as they enter the facility to prevent putting their lives in danger. The screening can be accomplished in the preparticipation physical. The purpose of this screening is to provide medical clearance for participation in all aspects of competitive

sport by using systematic evaluation.²³ These evaluations will check orthopedic stability, past history of injury or illness and will give a sketchy idea of cardiovascular health. A current debate in institutions all over the United States, however, argues whether or not athletes should have a comprehensive preparticipation cardiac screening before being cleared to participate. The goal of this type of screen would be to identify athletes with unsuspected heart disease that could predispose them to sudden cardiac death.¹

When designing a preparticipation cardiac screening, there are several items that must be kept in mind. First, screenings should be designed by taking into account that sudden cardiac death in athletes is very rare.²³ More than likely, the physician will not find any problems with the athlete. The physician should take a good medical history, including family history, history of chest pain, palpitations or irregular heart beats and syncope.¹ This cardiac screening should fit in with the rest of the preparticipation physical. Interestingly enough, universally accepted standards currently exist for this screening.²³ If the athlete's initial history and physical examination lead the physician to believe that a problem may be present, further diagnostic testing should be ordered. Diagnostic testing, history and physical examination, no matter how well they were performed, are not going to be able to catch every condition.²³ As with driving, there is never going to be a situation where there is no risk.²³ The athlete must realize that by participating in organized sports, there is always a chance for injury or even death.

The major limitation of the preparticipation cardiac screening is not the unwillingness of the physician to perform the tests, but rather the high cost of common cardiac screening procedures. The athletic population, especially at the college level, is relatively healthy. Most of the cardiac screens, therefore, would cost a great deal but not

yield any useful results.¹ Take, for example, the ECG. When given alone, the ECG is a fairly inexpensive test.¹ When a Division I university would perform the test on all of its athletes, however, the cost would quickly mount up. (Many large universities have as many as 800 athletes to screen!) Also, an ECG that shows an abnormality must be followed up with another test.¹ Since finding an abnormality is fairly common, the cost of screening every athlete skyrockets. The cost for ECG testing alone can go anywhere from \$400 to \$2,000 per test.²³ It is obvious to see why this type of screening would be impossible for schools with small budgets. Some larger schools (large Division I schools, for example) are able to get the procedures necessary for cardiac screening donated from a local hospital. The hospital exchanges its services and use of equipment for affiliation with the university. This is still very unique, however, and is not commonly seen.

Legally speaking, it is expected that in our society that the physician make a prudent effort to minimize as much risk as possible for the athlete.²³ When an athlete is examined by the physician, she expects the physician to perform her examination to the best of his ability. It is also expected that every athlete that is given a physical is given one of equal quality.²³ Because a comprehensive cardiac evaluation is usually too expensive to be performed, however, where is the line drawn for the amount of risk the physician is accountable for? As Maron, Thompson, Puffer and others state, the physician who has provided cardiac clearance for an athlete to participate in athletics is not liable for any undiscovered cardiac condition. For the athlete to win a malpractice suit against the physician, she would have to prove that the physician deviated from the customary practice that any prudent medical professional would have used.²³ In other

words, if the physician performed the athlete's screening according to the standard of care established by the facility where he is performing the screen, he is not liable. It is important the physician be thorough in their examinations and pay close attention to every detail to protect the athlete and himself.

Treatment Options

When an athlete is diagnosed with neurocardiogenic syncope, the Athletic Trainer and the physician should do everything within their scope of practice to keep the athlete in competition. Neurocardiogenic syncope can be very hard to treat, however, and not every athlete will be able to fully return to participation.⁷ The basis of all treatment protocols is the idea that the triggering mechanisms for neurocardiogenic syncope episodes can be prevented.⁵ Many episodes can be prevented by knowing the signs and symptoms of the prodrome and using common sense to avoid a complete syncopal episode. The goal of a treatment program should be to find a way to improve the quality of the athlete's life. Treatment protocols aim at avoiding as many recurrences as possible.²⁴ When there are less recurrences, the athlete does not have to worry about syncope so much and can spend time on other things. This improves the quality of the athlete's life. As stated before, however, treatment is not easy. Each protocol must be tailored to fit the athlete and adjusted until the right solution is found.²

One option for treatment is the use of pharmacotherapy.¹⁴ This treatment option is chosen often and is very popular. It is easy to get caught up in the use of drugs as a cure-all solution, however. Most guidelines, on the other hand, suggest that pharmacotherapy is required to stop recurring episodes that are disrupting normal daily

activity.¹⁴ The most popular classification of drugs used to prevent syncopal episodes are beta-adrenergic blocking agents.²² Beta-blockers reduce the output of the heart at rest and during exercise.²⁵ They reduce exercise heart rate and blood pressure.²⁵ In short, these drugs are able to decrease the workload of the heart both at rest and during exercise. It is the goal of these drugs to make it easier for the heart to keep up with the body during exercise, preventing decreased venous return.²² Not every athlete responds well to beta-blockers, however. Older athletes commonly have better responses to beta-blockade.²⁶ In some patients, these drugs can actually make the condition worse.²⁶ The side effects of these drugs are also hard for some athletes to bear. Beta-blockers are notorious for making the athlete feel lathargic and nauseous, both of which can negatively affect performance.²⁵ The drugs are also known to cause bradycardia at rest, which is bothersome and uncomfortable for some athletes.²⁷ The most important side effect to be aware of, however, is that beta-blocking agents can mask the symptoms of low blood sugar and must never be prescribed to diabetic athletes.^{25,27} Two commonly used beta-blockers are Atenolol and Pindolol.²⁵

There are several other drug classifications that can be used to treat neurocardiogenic syncope. Some physicians will choose to use antiarrhythmic agents, which are used to help control or prevent irregular heart beat patterns.²⁵ A commonly used antiarrhythmic drug is Norpace.²⁵ To understand the pharmacology, however, one must be well versed in how several classifications of drugs work and how they all react together. It also has been found in many cases that a combination of cardiac drugs is effective in small doses, instead of using one drug at a high dose.² It is the job of the

cardiologist or physician to know the medications, their dosage levels, indications and contraindications so a safe balance can be found for each individual athlete.

It is important that the physician monitor the athlete closely when using drugs to control syncope. He must watch how the athlete is responding to the drugs and constantly adjust the prescriptions to match the needs of the patient. In most cases, it is recommended that pharmacotherapy be discontinued after one year.³ If the athlete's condition worsens after ending treatment, however, they should be returned to the drug. If there is no change, drug therapy can be discontinued permanently. The prescribing physician must also be aware that some drugs that are used for treating neurocardiogenic syncope are listed as being banned by the NCAA.⁷ These drugs must be avoided if the athlete is going to continue participation. Contrary to popular belief, most syncope drugs are not banned due to their ability to enhance performance, but rather their ability to blunt performance.⁷ For example, the athlete who is lethargic from taking a beta-blocker will not be able to respond as quickly as normal, which may lead to injury.

A new and somewhat controversial alternative to treatment has been noted in recent research. This treatment involves repeated and prolonged exposure of the body to gravitational stress, much like that which is experienced during a tilt-table test.²⁸ This treatment subjects the athlete to repeated tilt-table testing. At each visit, the test is performed until the 45 minute time limit is up, or the athlete has an episode of syncope.²⁸ When not in the laboratory, the subjects were instructed to lean their upper backs against a wall for 30 minutes twice daily.²⁸ Standing in this position is supposed to simulate a tilt-table situation.²⁸ The last part of this treatment involved sleeping in a head-up tilted bed (using extra pillows, for example).²⁸ The idea behind this treatment is to attempt to

continually expose the athlete to situations in which the orthostatic balance is compromised.²⁸ It is believed that this repeated exposure will help the body “get used to” the condition and lessen the chances of a dramatic systemic reaction. If the body responds less dramatically, the athlete will have less episodes of syncope. In fact, in a study done by Ector, Reybrouck, Heidbuchel and others, all subjects in the study ended up free of syncope without the use of any pharmacologic therapy. This treatment protocol can also be carried out without using multiple tilt-table tests. The therapy can simply be performed at home against a wall, using another person to help support the participant should she experience syncope.²⁸

Another controversial treatment for neurocardiogenic syncope is permanent atrioventricular pacing.⁵ This involves putting a pacemaker in the heart and using it to control the heart’s rhythm.⁵ Amazingly, however, most studies show that this treatment has little or no ability to prevent neurocardiogenic syncope. In fact, in one study, 21 out of 22 consecutive patients who showed significant bradycardia during a tilt-table test had no success with permanent pacing.⁵ Pacing is a very invasive treatment, and is a treatment that will effect the patient for the rest of his or her life. It should only be considered as a last resort.⁵ More research on pacing needs to be done, however, to determine its true effectiveness. Most researchers agree that the benefits (or drawbacks) of pacing have not been determined. The success of pacing is also very patient specific, so the cardiologist and the patient must both agree to the procedure.

One of the most important aspects of any treatment protocol for neurocardiogenic syncope is to educate the patient about the condition. Calkins feels that educating the athlete can not be underestimated. He suggests teaching the athlete situations in which

neurocardiogenic syncope is most likely to occur and how to avoid them. He also suggests teaching the athlete the importance of lying down as soon as she senses prodromal symptoms.³ As stated earlier, if the athlete can learn to use common sense, many episodes can be avoided. In many cases, the cardiologist performing the athlete's first tilt-table test will provide a good deal of education to the athlete. For this reason, many athletes will see a great deal of improvement shortly after the test.⁴ It could also be that the athlete is reassured by the cardiologist who performs the test. He or she will know a great deal about the condition and will be able to share this information with the athlete.¹⁰ Some studies have even shown that not treating the athlete at all may be more effective than any other method of treatment.⁴ Treating the athlete with education only involves less risk, but may not be practical for the intercollegiate setting.

The athlete with neurocardiogenic syncope also needs to be educated on how to lead a healthy lifestyle. Many athletes, especially those at the collegiate level do not know how (or do not take the proper time) to take care of their bodies. They do not get enough sleep, consume too much alcohol and do not eat healthy diets. It is suggested that the athlete with neurocardiogenic syncope should eat a healthy diet and increase sodium intake to at least 3 grams per day.³ The athlete's syncopal condition and their general health will improve with a little counseling. On the collegiate level, the Athletic Trainer can provide a great deal of support to the athlete in this area. In fact, many college Athletic Trainers have access to the services of nutritionists and psychologists that may be able to assist the athlete in forming good habits. This education in supplement to other treatment may help the athlete wash neurocardiogenic syncope out of her daily life.

Giving the athlete confidence that she can overcome the condition may also be one of the most powerful tools the Athletic Trainer or physician may be able to give the athlete.

Conclusion

Neurocardiogenic syncope is a fairly new condition in intercollegiate athletics. The Athletic Trainer, team physician and coach must be constantly aware of the health and safety of the neurocardiogenic syncopal athlete. The Athletic Trainer must have a working knowledge of the anatomy and physiology of the cardiovascular system and its components. He or she must also know the warning signs and symptoms associated with a syncopal episode. At times, being able to make the correct referrals is one of the most important skills an Athletic Trainer can possess. Knowing the signs and symptoms of syncope can be the key for the Athletic Trainer to recognize a serious condition that needs to be referred. A working knowledge of the diagnostic testing and treatments of neurocardiogenic syncope can allow the Athletic Trainer to become more involved with the treatment of the athlete.

On the intercollegiate level, the Athletic Trainer must be aware of any and all conditions that may slow the athlete down. Especially on the Division I level, the Athletic Trainer will be held at least partially accountable for the athlete's playing status. Knowing the condition, the limitations that the athlete will experience, and ways to modify performance and life style will allow the Athletic Trainer to give the athlete the best health care available. The purpose of this examination was to explain the presentation, recognition, anatomical and physiological concerns, prodrome, diagnostic techniques and testing, effects on daily living, preparticipation screening and treatment

options associated with neurocardiogenic syncope. This examination, however, only “scratches the surface.” The following case study will also provide a realistic example of the limitations and possible clinical course of the neurocardiogenic syncopal athlete. Hopefully these tools can be utilized to prevent fear of a virtually benign condition, provide knowledge and stimulate research that will benefit future athletes.

Neurocardiogenic Syncope in a Collegiate Basketball

Player:

A Case Study

Personal Data:

This case presents a 19 year old female intercollegiate basketball player. The athlete has noted no previous history of physical problems according to her pre-participation physical exam. She is apparently healthy and has no history of heart disease in her family. At the onset of the illness, she was in her first season of intercollegiate participation.

Physical Signs and Symptoms:

The athlete first presented with symptoms while lifting weights during a pre-season conditioning session for the 1998-99 competitive season. Her symptoms included lightheadedness, diaphoresis, nausea, increased heart rate and pallor. The athlete described feeling as if she was going to faint, although a full syncopal episode was not attained. After the episode, the athlete complained of feeling “groggy” and very tired. She did not state being confused, and she was aware of her surroundings. This condition continued for approximately 20 minutes. After this time, she still felt tired, but was able to return to her residence. She did state that she had experienced this type of episode during her high school career, which was not noted on her pre-participation physical.

As pre-season conditioning continued, the athlete proceeded to have episodes of pre-syncope with much the same prodrome. She eventually experienced a full syncopal episode while running on an outdoor track. The athlete was fully unconscious for a period of about one minute, and then semi-conscious for approximately an additional two to three minutes. Again, the athlete experienced diaphoresis, lightheadedness, a racing heart rate, and other aspects of the prodrome that she had experienced during the presyncopal episodes. Also noted by the Athletic Trainer was a period of post-syncopal

fatigue. The fatigue lasted for at least 20-30 minutes before the athlete felt well enough to go home.

As the season progressed, the athlete continued to have episodes. At times, the Athletic Trainer or the athlete could recognize the prodrome before the episode. If this was the case, the athlete could be strategically positioned to prevent a full syncopal event. She was unable to compete or participate in any activity fully, and required a longer rest period than the other athletes between activities. She had episodes during exercise, after exercise, when under stress or experiencing emotional challenges, when in pain and at the sight of blood, to name a few precursors. During each episode, the Athletic Trainer noted an increase in heart rate as the episode started which fell very quickly as the condition progressed. The athlete could also be seen checking her pulse at the carotid artery or shaking her hands (a sign that her hands were “tingling” due to lack of blood flow) before each episode could occur. The Athletic Trainer and coaching staff had to be constantly aware of the athlete’s presence in an attempt to prevent secondary injury from loss of consciousness.

Differential Diagnosis:

After the first couple presyncopal episodes and the discovery of the athlete’s previous history of episodes, the athlete was referred to a team physician and a cardiologist. After a great deal of testing, the athlete was diagnosed with neurocardiogenic syncope. This diagnosis could not be made until all other conditions and abnormalities were ruled out. The physicians and cardiologists were careful to rule out underlying cardiac abnormalities, arrhythmia and electrical conduction problems of the heart and cardiovascular system.

Results of Diagnostic Imaging/Laboratory Tests:

Testing for heart abnormalities was first performed by the athlete's cardiologist. The athlete was subjected to a battery of tests including an ECG, echocardiogram and a treadmill stress test. All of these tests showed no cardiac problems or heart abnormalities, so the athlete was cleared to play. When the symptoms continued to worsen, however, the athlete was referred for a tilt-table test. This test was the only tool able to reproduce the athlete's symptoms. Not only did the athlete have a full syncopal episode during the test, but was recorded as reaching a period of asystole. This test confirmed the diagnosis of neurocardiogenic syncope.

With the diagnosis confirmed, the athlete was subjected to several other tests to attempt to find an effective treatment. The most significant of these tests was the implantation of a subcutaneous memory loop recorder. The device was placed in the athlete's upper left chest and was accompanied by a "beeper" that served as the activating device. The athlete was to wear the device at all times and was to activate the recorder during an episode. The recorder was capable of recording an ECG reading for a period 12 minutes before activation and a few minutes after.

Clinical Course:

As stated earlier, this athlete had to be constantly monitored. She could have an episode at any time. She was first placed on Atenolol (a Beta-Blocker) and her activity was monitored. There were attempts to modify her caffeine intake, sodium intake and to improve her diet. None of these treatments seemed to help, however, and the condition continued. Despite readings from the loop recorder and attempts to change her medications, the athlete did not see much improvement. She attempted to continue with

another year of intercollegiate participation, but was unsuccessful. She eventually chose to leave the team in an attempt to get a better control over her syncope. The athlete did have a period where the condition appeared to be improving, but she continues to have problems at the present time. She states that the condition “comes and goes” for extended periods. Her cardiologist is also considering the use of permanent pacing to attempt to control the condition.

Deviation from Expected:

There are several deviations from the normal with this athlete. First of all, she has not seen any real improvement in her condition, despite education and treatment. It has been approximately one and a half years since her diagnosis, and her doctor is now considering pacing. Despite all attempts to adjust the athlete’s nutrition, lifestyle and medication, she continues to have episodes. At this time, the athlete is very frustrated with the condition and states that she will try almost anything if she could stop the episodes.

What is also important to note in this case is the fact that the athlete did not note any problems or abnormalities involving syncope on her preparticipation physical upon arriving at the university. It is unknown if a preparticipation cardiac screening would have caught the condition. In fact, a cardiac screen would have included an ECG and an echocardiogram, both of which were negative in this athlete. This case illustrates that it is imperative that the athlete notifies the Athletic Training staff of any pre-existing conditions. This could have lead to a more timely diagnosis of the athlete’s syncope.

The most important fact to note in this case, however, is that the athlete is no longer able to participate in intercollegiate athletics. Her attempts to do so were

unsuccessful, however, due to her decreased ability to meet the physical demands necessary to perform on the Division I level. The athlete is able to play on her own at this time, but has trouble finishing a game. Despite a great deal of research stating that neurocardiogenic syncope patients can participate in athletics, it is case specific. This athlete can only participate at about half of her normal capacity. The athlete does deserve credit for giving her best effort, but to play on the intercollegiate level with her amount of disability was not feasible. The quality of life for this athlete has greatly decreased, and her amount of morbidity often keeps her from living the type of life she would like to. There is hope that she will be able to find a treatment to control her condition, but she will most likely live with it for the rest of her life.


References

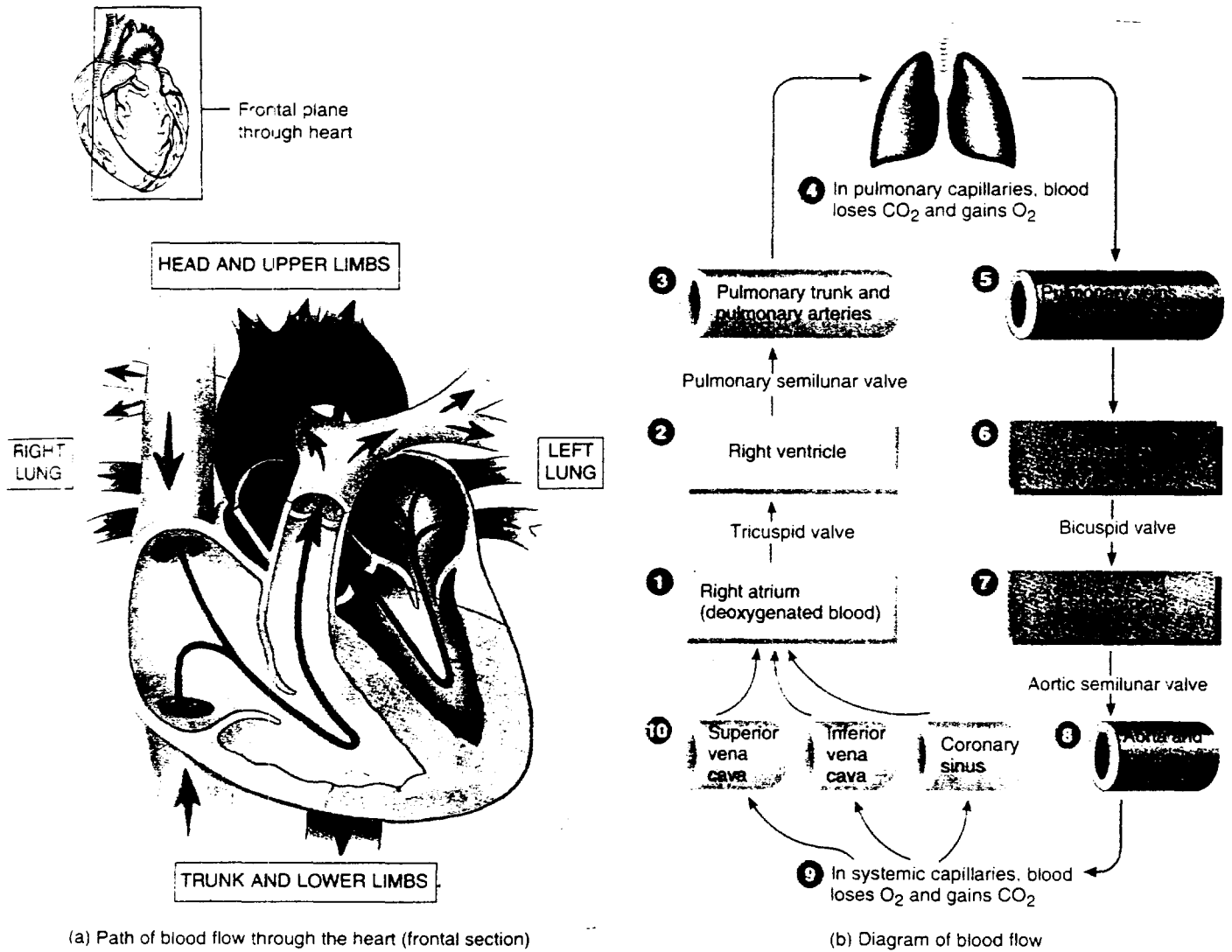
1. Basilico FC. Cardiovascular disease in athletes. *Am J Sports Med.* 1999;27(1):108-121.
2. Grubb BP, Karas B. Diagnosis and management of neurocardiogenic syncope. *Current Opinion in Cardiology.* 1998;13:29-35.
3. Calkins H. Understanding neurocardiogenic syncope in athletes. *J Musculoskel Med.* 1997;14(7):37-46.
4. Barbey J. Vasodepressor syncope diagnosis and management. *Cardiology Clinics.* 1997;15(2):251-256.
5. Sra JS, Jazayeri MR, Dhala A, and others. Neurocardiogenic syncope diagnosis, mechanisms, and treatment. *Cardiology Clinics.* 1993;11(1):183-191.
6. Shen WK. The fainting athlete: when is it a heart problem? *Your Patient & Fitness.* 1996;11(1):26j-26v.
7. Wang D, Sakaguchi S, Babcock M. Exercise-induced vasovagal syncope: limiting the risks. *Phys Sportsmed.* 1997;25(5):64-74.
8. Linzer M, Gold DT, Pontinen M, and others. Recurrent syncope as a chronic disease: preliminary validation of a disease-specific measure of functional impairment. *J General Internal Med.* 1994;9:181-185.
9. Calkins H, Seifert M, Morady F. Clinical presentation and long-term follow-up of athletes with exercise-induced vasodepressor syncope. *Am heart J.* 1995;129:1159-64.
10. Natale A, Geiger MJ, Maglio C, and others. Recurrence of neurocardiogenic syncope without pharmacologic interventions. *Am J Cardiology.* 1996;77:1001-3.
11. Kapoor WN. Evaluation and management of syncope. *Contemporary Internal Medicine.* 1994;6(11):29-39.
12. Tortora GJ, Grabowski SR. *Principles of Anatomy and Physiology.* 8th ed. New York, NY: Harper Collins College Publishers;1996:553-667.
13. Grubb BP, Kosinski D. Dysautonomic syncope. *Cardiology Clinics.* 1997;15(2):257-268.
14. Grubb BP, Kimmel S. Head-upright tilt table testing: a safe and easy way to assess neurocardiogenic syncope. *Postgraduate Medicine.* 1998;103(1):133-140.
15. Kaufmann H. Syncope: a neurologist's viewpoint. *Cardiology Clinics.* 1997;15(2):177-194.
16. Fischer R. *Biopsychology.* Muncie, IN: Tichenor Publishing;1999:155-164.
17. Benke T, Hochleitner M, Bauer G. Aura phenomena during syncope. *Eur Neurol.* 1997;37:28-32.
18. Tanel RE, Walsh EP. Syncope in the pediatric patient. *Cardiology Clinics.* 1997;15(2):277-294.
19. Linzer M, Pontinen M, Gold D, and others. Impairment of physical and psychosocial function in recurrent syncope. *J Clin Epidemiol.* 1991;44(10):1037-43.
20. Hammill SC. Value and limitations of noninvasive assessment of syncope. *Cardiology Clinics.* 1997;15(2):195-218.
21. Benditt DG, Lurie KG, Fabian WH. Clinical approach to diagnosis of syncope: an overview. *Cardiology Clinics.* 1997;15(2):165-176.

22. Mahanonda N, Bhuripanyo K, Kangkagate C, and others. Randomized double-blind, placebo-controlled trial of oral atenolol in patients with unexplained syncope and positive upright tilt table test results. *Am Heart J*. 1995;130(6):1250-53.
23. Maron BJ, Thompson PD, Puffer JC, and others. Cardiovascular preparticipation screening of competitive athletes. *Circulation*. 1996;94:850-856.
24. Ruiz GA, Peralta A, Gonzalez-Zuelgaray J, and others. Evolution of patients with clinical neurocardiogenic (vasovagal) syncope not subjected to specific treatment. *Am Heart J*. 1995;130(2):345-350.
25. Martin M, Yates W. *Therapeutic Medications in Sports Medicine*. Baltimore, MD: Williams & Wilkins; 1998:81-82.
26. Natale A, Newby K, Dhala A, and others. Response to beta blockers in patients with neurocardiogenic syncope: how to predict beneficial effects. *J Cardiovasc Electrophysiol*. 1996;7:1154-8.
27. Iskos D, Dutton J, Sheinman MM, and others. Usefulness of pindolol in neurocardiogenic syncope. *Am J Cardiology*. 1998;82:1121-24.
28. Ector H, Reybrouck T, Heidbuchel H, and others. Tilt training: a new treatment for recurrent neurocardiogenic syncope and severe orthostatic intolerance. *PACE*. 1998;21:193-196.
29. Miles WM. Driving issues related to arrhythmic syncope. *Cardiology Clinics*. 1997;15(2):327-339.

Appendix A:
Blood Flow Through the Human Heart

Figure 20.6 Blood flow through the pulmonary and systemic circulations.

 Blood flowing through lung capillaries loses CO_2 and gains O_2 ; blood flowing through systemic capillaries loses O_2 and gains CO_2 .

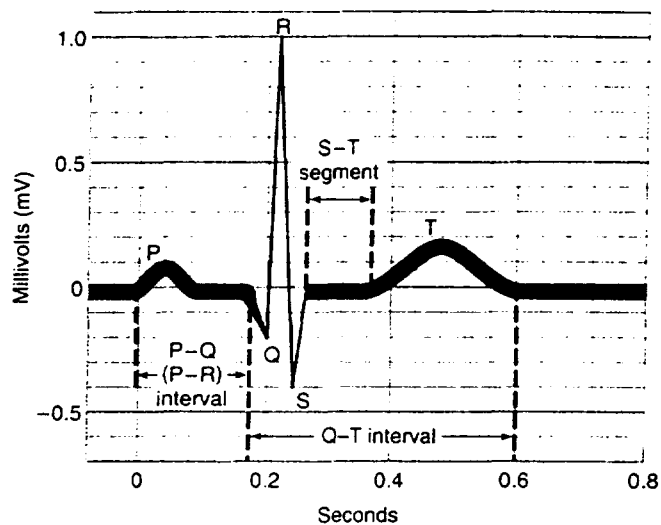


Appendix B:
A Normal ECG Recording

Figure 20.10 Electrocardiogram or ECG (Lead II).



An ECG is a recording of the electrical activity that accompanies each heartbeat.



Normal ECG of a single heartbeat

MEANING OF ECG WAVES

P wave = atrial depolarization

QRS complex = onset of ventricular depolarization

T wave = ventricular repolarization

Appendix C:
Tilt Table Testing

Figure 1

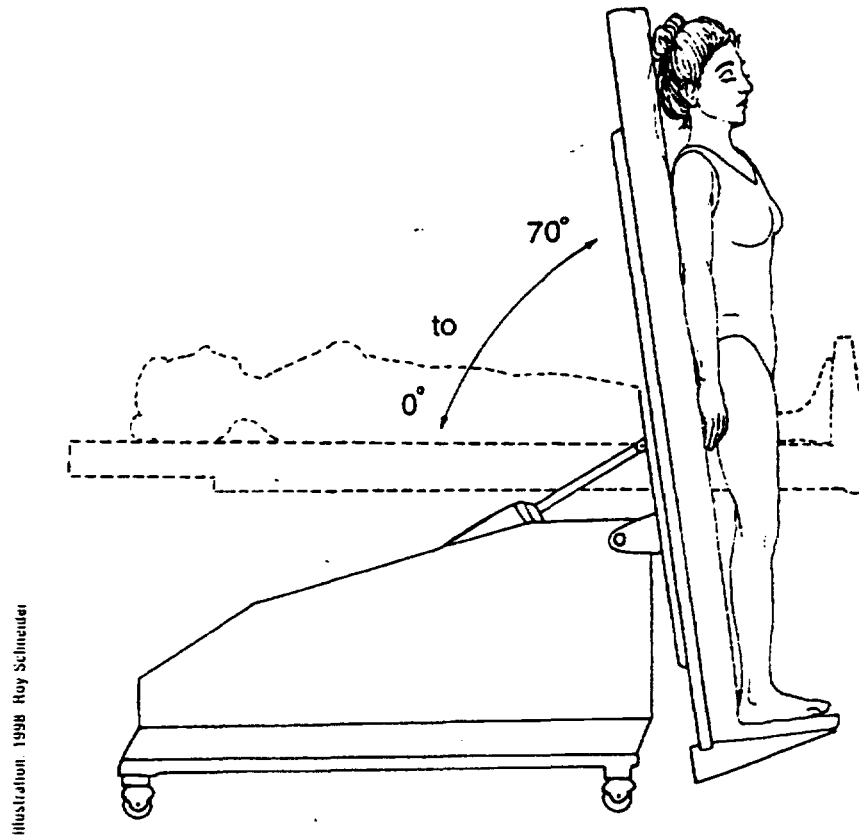
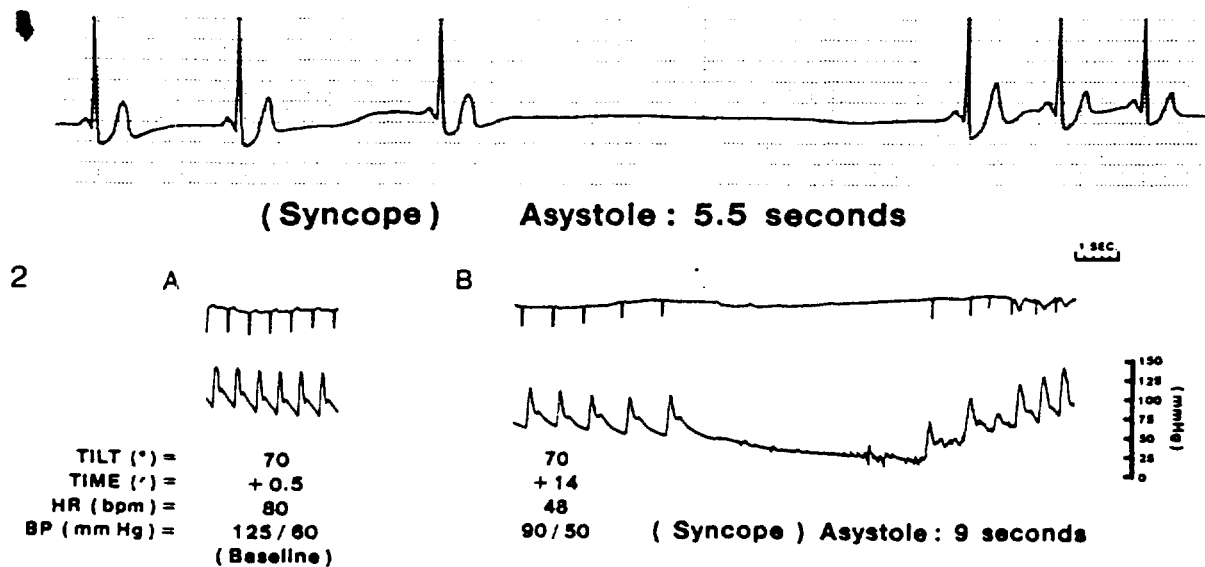


Figure 1. Head-upright tilt table testing. Procedure begins with patient supine, and table is slowly tilted upright to 70° angle.

Figure 2



1. Grubb BP, Kimmel S. Head upright tilt table testing. *Postgraduate Medicine*. 1998;103(1):136.
2. Sra JS, Jazayeri MR, Dhala A, and others. Neurocardiogenic syncope: diagnosis, mechanisms, and treatment. *Cardiology Clinics*. 1993;11(1):188.

Appendix D:
Possible Risk Involved in Popular Sports

TABLE 4
Types of Exercise by Sport*

	A. Low dynamic	B. Moderate dynamic	C. High dynamic
I. Low static	Billiards Bowling Cricket Curling Golf Riflery	Baseball Softball Table tennis Tennis (doubles) Volleyball	Badminton Cross-country skiing (classic technique) Field hockey* Orienteering Race walking Racquetball Running (long distance) Soccer* Squash Tennis (singles)
II. Moderate static	Archery Auto racing*† Diving*† Equestrian*† Motorcycling*†	Fencing Field events (jumping) Figure skating* Football (American)* Rodeoing*† Rugby* Running (sprint) Surfing*† Synchronized swimming†	Basketball* Ice hockey* Cross-country skiing (skating technique) Football (Australian rules)* Lacrosse* Running (middle distance) Swimming Team handball
III. High static	Bobsledding*† Field events (throwing) Gymnastics*† Karate/judo* Luge*† Sailing Rock climbing*† Waterskiing*† Weight lifting*† Windsurfing*†	Body building*† Downhill skiing*† Wrestling*	Boxing* Canoeing/kayaking Cycling*† Decathlon Rowing Speed skating

* Danger of bodily collision.

† Increased risk if syncope occurs.

* Reprinted with permission from the American College of Cardiology (Mitchell et al., *J Am Coll Cardiol* 28: 845-899, 1994).

Appendix E:
Syncope Functional Status Questionnaire

Syncope Functional Status Questionnaire

Last Name	First Name	Hospital ID																
<p>I) Please answer the following as: Y for yes, N for no, or N/A for non-applicable. These episodes affect my life and daily routine by:</p> <p>() interfering with my life or routine</p> <p>() preventing or causing me to avoid driving a vehicle</p> <p>() reducing the amount of walking I do each day</p> <p>() interfering with my use of public transportation (buses, trains, etc.)</p> <p>() interfering with my performing errands (grocery shopping, housekeeping)</p> <p>() interfering with my physical activities (entertainment, sports)</p> <p>() affecting my ability to work at my job</p> <p>() affecting my relations with my spouse/boyfriend/girlfriend</p> <p>() affecting my relationship with my family</p> <p>() affecting my relationships with my friends</p> <p>() affecting my sexual functioning</p>																		
<p>II) How much do you worry about your episodes? On the scale below, circle the number that fits you.</p> <table style="width: 100%; border: none;"> <tr> <td style="text-align: center;">1</td> <td style="text-align: center;">2</td> <td style="text-align: center;">3</td> <td style="text-align: center;">4</td> <td style="text-align: center;">5</td> <td style="text-align: center;">6</td> <td style="text-align: center;">7</td> <td style="text-align: center;">8</td> </tr> <tr> <td colspan="7" style="text-align: left;">All I do is worry</td> <td style="text-align: right;">I never worry</td> </tr> </table>			1	2	3	4	5	6	7	8	All I do is worry							I never worry
1	2	3	4	5	6	7	8											
All I do is worry							I never worry											
<p>III) How much do you fear a typical episode coming on? On the scale below, circle the number that fits you.</p> <table style="width: 100%; border: none;"> <tr> <td style="text-align: center;">1</td> <td style="text-align: center;">2</td> <td style="text-align: center;">3</td> <td style="text-align: center;">4</td> <td style="text-align: center;">5</td> <td style="text-align: center;">6</td> <td style="text-align: center;">7</td> <td style="text-align: center;">8</td> </tr> <tr> <td colspan="3" style="text-align: left;">I am terrified</td> <td style="text-align: center;">neutral</td> <td colspan="4" style="text-align: right;">I have no fear</td> </tr> </table>			1	2	3	4	5	6	7	8	I am terrified			neutral	I have no fear			
1	2	3	4	5	6	7	8											
I am terrified			neutral	I have no fear														
<p>IV) How does worry about an episode affect your daily life? On the scale below, circle the number that fits you.</p> <table style="width: 100%; border: none;"> <tr> <td style="text-align: center;">1</td> <td style="text-align: center;">2</td> <td style="text-align: center;">3</td> <td style="text-align: center;">4</td> <td style="text-align: center;">5</td> <td style="text-align: center;">6</td> <td style="text-align: center;">7</td> <td style="text-align: center;">8</td> </tr> <tr> <td colspan="5" style="text-align: left;">All I do is worry, so worry totally keeps me from my routine</td> <td colspan="3" style="text-align: right;">I never worry, so worry does not interfere</td> </tr> </table>			1	2	3	4	5	6	7	8	All I do is worry, so worry totally keeps me from my routine					I never worry, so worry does not interfere		
1	2	3	4	5	6	7	8											
All I do is worry, so worry totally keeps me from my routine					I never worry, so worry does not interfere													

Directions for scoring the Syncope Functional Status Questionnaire:

The Impairment Score is computed as the percentage of applicable areas out of 11 for which the patient reports that syncope affects him or her. For instance, if a patient is unemployed, making the "work at job" item not applicable, the score would be based upon ten items. If the patient answered yes to three of those ten, his or her score would be 30, or 30%. The Fear/Worry Score is based upon the average of the three Likert items after they are rescaled to range from 100 to 0 instead of 1 to 8.* For instance, if the patient answers 2, 6, and 3 to the three questions, respectively, these would be rescaled to 85.7, 14.3, and 71.4, and averaged to give a Fear/Worry Score of 61.9. (Note that it would be more efficient to average the three Likert values before rescaling.)

The Syncope Dysfunction Score (SDS) is the average of the Impairment Score and the Fear/Worry Score. For the hypothetical patient noted above, the SDS would be 46.

*The rescaling formula is:

$$\frac{100 \times (8 - \text{Likert value})}{7}$$

Linzer M, Gold DT, Pontinen M, and others. Recurrent syncope as a chronic disease. *J Gen Intern Med.* 1994;9:186.